Intensive Care Med (2006) 32:836–842
DOI 10.1007/s00134-006-0178-6

ORIGINAL

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Amplitude-integrated EEG (aEEG) predicts outcome after cardiac arrest and induced hypothermia

Received: 31 January 2006
Accepted: 31 March 2006
Published online: 29 April 2006

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Abstract *Objective:* To evaluate the use of continuous amplitude-integrated EEG (aEEG) as a prognostic tool for survival and neurological outcome in cardiac arrest patients treated with hypothermia. *Design:* Prospective, observational study. *Setting:* Multidisciplinary intensive care unit in a university hospital. *Intervention:* Comatose survivors of cardiac arrest were treated with induced hypothermia for 24 h. An aEEG recording was initiated upon arrival at the ICU and continued until the patient regained consciousness or, if the patient remained in coma, no longer than 120 h. The aEEG recording was not available to the ICU physician, and the aEEG tracings were interpreted by a neurophysiologist with no knowledge of the patient's clinical status. Only clinically visible seizures were treated. *Measurements and results:* Thirty-four consecutive hypothermia-treated cardiac arrest survivors were included. At normothermia (mean 37 h after cardiac arrest), the aEEG pattern was discriminative for outcome. All 20 patients with a continuous aEEG at this time regained consciousness, whereas 14 patients with pathological aEEG patterns (flat, suppression-burst or status epilepticus) did not regain consciousness and died in hospital. Patients were evaluated neurologically upon discharge from the ICU and after 6 months, using the Cerebral Performance Category (CPC) scale. Eighteen patients were alive with a good cerebral outcome (CPC 1–2) at 6-month follow-up. *Conclusion:* A continuous aEEG pattern at the time of normothermia was discriminative for regaining consciousness. aEEG is an easily applied method in the ICU setting.

Keywords Cardiac arrest · Coma · Hypothermia · aEEG · Outcome

Introduction

Outcome after cardiac arrest is poor and has not improved over recent decades [1]. Most patients with return of spontaneous circulation (ROSC) are in deep coma and are treated in the intensive care unit (ICU). Early prediction of neurological outcome is essential so that relevant care can be given to patients awakening from coma, and futile intensive care be avoided in patients with severe hypoxic ischaemic encephalopathy (HIE). Several prognostic measures have been evaluated [2], including biochemical markers in peripheral blood [3, 4, 5], electroencephalogram (EEG) [6, 7], somatosensory evoked potentials (SSEP) [8] and combinations thereof [9, 10]. However, a clinical neurological examination is still the most widely used and best validated prognostic measure [11, 12, 13, 14]. The introduction of therapeutic hypothermia to treat comatose cardiac arrest survivors has improved the outcome [15, 16], and re-evaluation of our prognostic tools is necessary.

The EEG pattern after anoxic coma has been studied in animal models [17, 18, 19] and also in humans [6, 7]. Early recovery of cortical activity is correlated with a good outcome, whereas the occurrence of seizures post

arrest is associated with a poor outcome [20]. Treating post-anoxic seizures seems reasonable, but whether or not this improves long-term outcome is less apparent, since seizures may be secondary to irreversible tissue damage. Amplitude-integrated EEG monitoring (aEEG) has been used to predict the outcome in asphyctic neonates [21, 22, 23], and recently aEEG was used to provide early objective information about the preceding injury in neonates treated with induced hypothermia [24]. In the present study, 34 consecutive comatose, hypothermia-treated cardiac arrest patients were monitored with aEEG in order to investigate its value as a prognostic tool for survival and long-term neurological outcome.

Materials and methods

This study was performed in a Swedish university hospital from January 2004 to February 2005. It was approved by the Regional Ethics Review Board in Lund and informed consent was obtained from the next of kin, or retrospectively from the patient. All adult comatose patients after cardiac arrest with a Glasgow Coma Scale (GCS) score of less than 8 who were eligible for therapeutic hypothermia were included. According to our protocol, comatose survivors are treated with hypothermia regardless of the initial cardiac rhythm or the location of arrest. Patients are excluded from treatment if cardiac arrest is secondary to aortic dissection, intracranial or massive bleeding or if the patient is terminally ill. In this study, as in clinical routine, hypothermia was induced using cold saline, 30 ml/kg, 4°C [25] and maintained using an external cooling device (CritiCool®, TREM Ltd., Israel or Arctic Sun®, Medivance Inc., CO, USA) or intravenous cooling (Icy Cath®, Alsius Corp., CA, USA). The patient's core temperature was measured in the bladder and maintained at 33.0 ± 1°C for 24 h.

Sedation was achieved prior to induction of hypothermia using fentanyl (1–2 μg/kg) and midazolam (0.05 mg/kg), and maintained in the ICU using fentanyl (1–2 μg/kg/h) and propofol (2–4 mg/kg/h) during hypothermia treatment. A nondepolarising muscle relaxant (rocuronium) was used intermittently to avoid compensatory shivering. Patients were evaluated by a cardiologist, and those eligible for invasive procedures were transported to the catheterisation laboratory for evaluation and treatment en route to the ICU.

On arrival at the ICU, a Nervus monitor® (Viasys Healthcare, WI, USA) was applied by the ICU nursing staff. The monitor recorded EEG from two bipolar EEG channels (C3–P3, C4–P4 according to the 10–20 system). Four subcutaneous needle electrodes were placed at central and parietal skull positions on each side, in addition to one combined electrode functioning as reference and ground. The raw EEG was displayed digitally on a screen (0.5–30 Hz, 1 s/div, 10 div/screen), and the aEEG (2–15 Hz, 4 h/screen) was displayed on a semi-logarithmic scale (0–100 μV). All EEG data were linked on-line to the Department of Neurophysiology and stored for further analysis. aEEG monitoring was discontinued when patients showed signs of awakening, at death, or, in cases of persistent coma, no later than 120 h post arrest. The attending ICU physician was not given access to the aEEG. Clinical seizures were noted and treated, using propofol alone or in combination with midazolam and/or fosfenytoin, with the intention of stopping visible seizures. The neurophysiologist (I.R.) had no knowledge of the clinical status of the patients at the time of evaluating the aEEGs. The initial aEEG pattern during the first 4 h of recording, and that for 4 h at the time the patient had regained normothermia, were evaluated and related to patient outcome. The aEEG patterns were classified into the following categories: extremely low voltage (flat; maximum voltage < 5 μV); discontinuous suppression-burst pattern; electrographic status epilepticus with recurrent epileptiform activity; and continuous EEG (Fig. 1a–d).

Patients were normoventilated and p-glucose was maintained at 5–8 mmol/l. Mean blood pressure above 65 mmHg and central venous oxygen saturation above 70% were aimed at, and when needed, vasoactive drugs (norepinephrine) and inotropic support (dobutamine or levosimendan) were administered. At the time of normothermia, sedation was reduced or withdrawn and the patients were evaluated neurologically. Agitated patients and patients not ready for extubation at this time (coma, fluid overload) were kept sedated at the lowest dose required to tolerate the endotracheal tube.

In patients not regaining consciousness, full supportive treatment was continued for at least 72 h after normothermia had been attained. At this time, a clinical neurological examination was performed in combination with bilateral somatosensory evoked potentials (SSEP). In patients with a GCS score of 3 or 4 and/or bilateral lack of cortical response, treatment was considered futile and active care was withdrawn. Patients regaining consciousness were extubated and evaluated neurologically before leaving the ICU, and again before leaving the hospital, using the five-grade Cerebral Performance Category (CPC) scale [26] (Table 1). A follow-up evaluation was performed by a neurologist 6 months after cardiac arrest. A CPC score

Table 1 Cerebral Performance Category (CPC) scale

CPC 1: Good cerebral performance: conscious, alert, able to work
CPC 2: Moderate cerebral disability: conscious, can carry out independent activities
CPC 3: Severe neurological disability: conscious, dependent on others for daily support
CPC 4: Coma or vegetative state
CPC 5: Dead

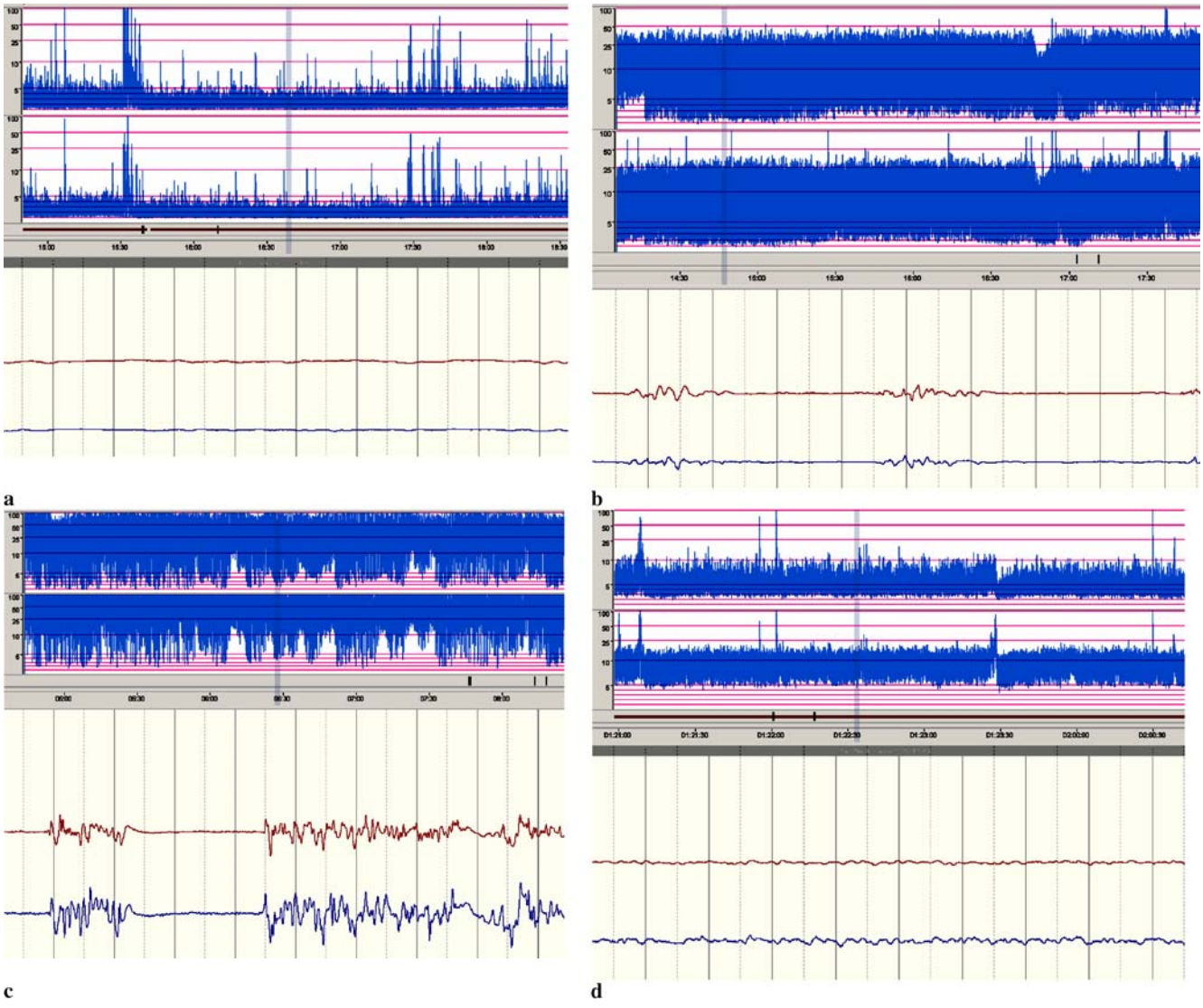

Fig. 1 Four common aEEG patterns in comatose survivors after cardiac arrest. **a** Flat EEG; **b** suppression-burst pattern; **c** electrographic status epilepticus; **d** continuous EEG

of 1–2 *at any time* during the 6-month follow-up period was considered a good neurological outcome, whereas a CPC score of 3–5 was considered a poor outcome.

Results

Thirty-four consecutive patients were included. Their mean age was 62 years, and 23 patients (68%) were male. The majority of the patients (82%) had suffered an out-of-hospital cardiac arrest. Twenty-one patients (62%) had an initial ventricular fibrillation (VF), while asystole and pulseless electric activity (PEA) were seen in seven patients and four patients respectively (20% and 12%). In two patients (6%) the initial cardiac rhythm was unknown. The mean time from cardiac arrest to the initiation of cardiac resuscitation by trained personnel was 7 min (range 1–16 min), and the mean time to ROSC was 20 min (range 2–60 min). GCS score at the initiation of hypothermia ranged from 3 to 7 (median 4). The target temperature was reached at 243 min (range 80–490 min), and normothermia was resumed at 37 h post arrest (range 30–40 h) (Table 2). EEG monitoring was commenced in the ICU between 2 h and 10 h after arrest and was technically successful in all patients.

The initial aEEG pattern was flat in 24 patients (71%), 7 patients (20%) showed a continuous aEEG, 2 patients (6%) showed a suppression-burst pattern, and 1 an alpha-coma pattern (3%) (Table 3). All patients with an initial continuous aEEG regained consciousness, whereas the

Table 2 Patient characteristics

Sex (no.)	
Male	23 (68%)
Female	11 (32%)
Age (years)	62 (29–84)
Initial rhythm (no.)	
VF	21 (62%)
Asystole	7 (21%)
PEA	4 (12%)
Unknown	2 (6%)
Location at time of cardiac arrest (no.)	
Out of hospital	28 (82%)
In hospital	6 (18%)
Time from collapse to	
CPR (min)	7 (1–16)
ROSC (min)	20 (2–60)
Target temp. (min)	243 (80–490)
Normothermia (h)	37 (30–40)
GCS score at initiation of treatment (median)	4 (3–7)

Numbers given as mean (range) unless otherwise indicated.
CPR, cardio-pulmonary resuscitation by medical personnel; ROSC, return of spontaneous circulation; GCS, Glasgow Coma Scale

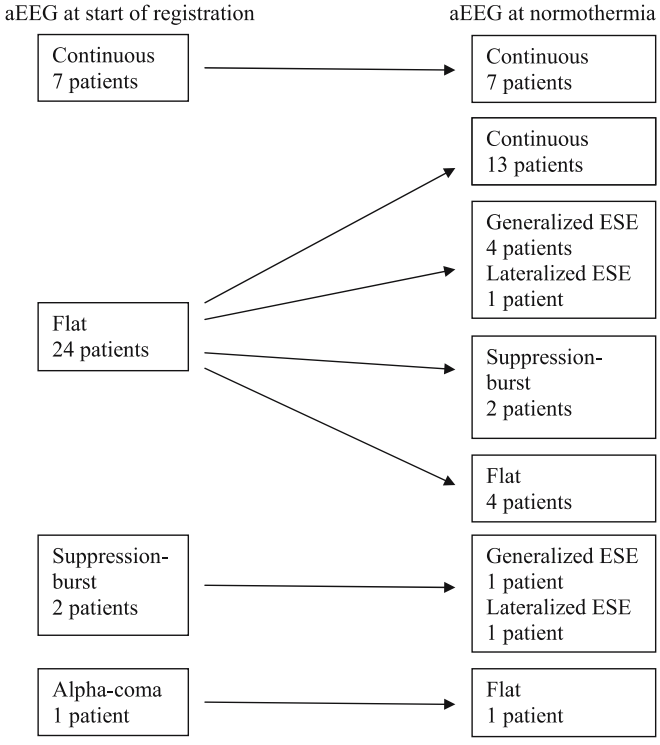

Fig. 2 Development of aEEG pattern from start of registration to normothermia in 34 comatose survivors after cardiac arrest (ESE, electrographic status epilepticus)

outcome for patients with an initially flat aEEG was mixed and inconclusive. The three patients exhibiting an initial suppression-burst or alpha-coma pattern did not regain consciousness and died in hospital.

The development of aEEG pattern from the start of registration until return to normothermia is presented in Fig. 2. At the time of normothermia, 20 patients had a continuous aEEG, all of whom regained consciousness (Table 3). At 6-month follow-up, a best CPC score of 1–2 (good outcome) was found in 18 out of these 20 patients, while one patient had a best CPC score of 3 and one patient (best CPC 3) had died due to cardiac failure. Four patients were contacted by telephone only, and no patient was lost to follow-up. All 14 patients with an aEEG pattern other than continuous at normothermia died during the hospital stay, without regaining consciousness (Table 3). Seven out of these 14 patients had a suppression-burst pattern or a flat aEEG, and the other seven patients had generalised (five patients) or lateralised (two patients) status epilepticus. The electrographic status epilepticus (ESE) started at a mean 18 h after cardiac arrest (range 9–29 h, median 10 h) and in all cases corre-

Table 3 The aEEG pattern versus best CPC during 6 months' follow-up

aEEG pattern	All patients	CPC 1	CPC 2	CPC 3	CPC 4
Initial					
Continuous	7	6	1	–	–
Suppression-burst	2	–	–	–	2
Alpha-coma	1	–	–	–	1
Flat	24	6	5	2	11
At normothermia					
Continuous	20	12	6	2[a]	–
Generalised status epilepticus	5	–	–	–	5[b]
Lateralised status epilepticus	2	–	–	–	2[b]
Suppression-burst	2	–	–	–	2[b]
Flat	5	–	–	–	5[b]

[a] One of two patients died of cardiac failure before leaving hospital.
[b] All patients died in hospital.

sponded to clinical seizures. Of the 14 patients who did not regain consciousness, three died due to circulatory collapse shortly after regaining normothermia (1–10 h). The remaining 11 patients were evaluated neurologically 72 h after normothermia had been attained; all had GCS values of 3 or 4. SSEP were performed in seven patients, five of whom showed no cortical response. Of the remaining two patients, one showed a bilateral response with a long latency in combination with a GCS 3 status. The other showed normal cortical SSEP responses, but widespread ischaemic damage on CT and MRI, and a GCS 4 status. Four patients were not examined using SSEP due to unavailability of a technician. Hence, in 11 patients intensive care was withdrawn and all died in hospital 4–11 days after cardiac arrest, without regaining consciousness.

No patient in our study had subclinical seizures until return to normothermia, at which time sedation was ongoing in 29 patients. All seven patients with clinical signs of seizure activity received propofol, at a mean dose of 1.4 mg/kg/h (range 0.5–3.7 mg/kg/h), and four patients received fentanyl infusion, mean 0.6 μg/kg/h (range 0.3–0.9 μg/kg/h). Four of the patients were additionally treated with midazolam, in two cases combined with fosfenytoin. Only transient effects on clinical seizures and ictal aEEG were observed.

Discussion

In this study, aEEG was investigated regarding its potential use as a predictor of outcome in hypothermia-treated comatose survivors of cardiac arrest. The presence of a continuous aEEG pattern at the start of registration correlated with a good outcome. Later, at the time of normothermia, the aEEG pattern was predictive of survival and neurological outcome in all patients. A continuous aEEG at this time correlated to regaining consciousness; all patients except one were alive 6 months later. A flat, suppression-burst or status epilepticus aEEG at the time of normothermia, however, was a strong predictor of a poor outcome, and all such patients died in hospital without regaining consciousness.

The initial aEEG was flat in the majority of patients, but over time most patients recovered aEEG activity (Fig. 2). At normothermia, 13 of 24 patients initially showing flat aEEGs presented a continuous aEEG, all of whom regained consciousness. The remaining 11 patients initially showing flat aEEGs presented various pathological aEEG patterns at normothermia (Fig. 2), and none regained consciousness. The inability of the initial EEG pattern to predict outcome in comatose survivors of cardiac arrest has been noted previously [7, 14], but those studies were performed before induced hypothermia was a therapeutic option. Our data from adult cardiac arrest patients are at variance with the findings in asphyctic neonates, where an initial flat, suppression-burst or low-voltage aEEG pattern (< 6 h) correlates with a poor outcome [24].

At normothermia, the aEEG pattern was discriminative regarding outcome in all our patients, supporting the view that EEG is most useful as a predictor of outcome 24 h or later after cardiac arrest [14, 27]. All 20 patients regaining consciousness showed a continuous aEEG at normothermia, 18 of whom were classified as having a good outcome. Fourteen patients with an aEEG other than continuous at the time of normothermia never regained consciousness (Table 3); all died in hospital 4–11 days after the arrest. Hence, no patients remained in a persistent vegetative state.

The core temperature was below 35°C in all patients at the start of aEEG registration and several patients had already reached the target temperature of 33± 1°C. In this temperature range (32–34°C) the EEG is not significantly affected by temperature, and the low temperature per se could not explain the high number of initial isoelectric aEEGs [28]. However, we cannot exclude a pharmacological effect on the initial aEEG, since all patients had received bolus doses of fentanyl and midazolam, and all were receiving a low-dose infusion of propofol. The combined anticonvulsive effect of hypothermia and the sedatives administered may have affected the recovery of aEEG and also the prevalence of seizures during hypothermia treatment. Still, seven patients (21%) developed clinical seizures and a corresponding ESE during hypothermia treatment. All seven received additional anticonvulsive therapy with only a transient effect on clinical seizures. None of them regained consciousness and all died during their hospital stay. Seizures due to HIE affected up to 40% of comatose cardiac arrest survivors in previous studies [20, 29]. It is not known whether treatment of seizures due to HIE affects the outcome or not, but it seems reasonable not to refrain from treatment until the matter has been further investigated.

There were no major differences in the aEEG pattern between the right and the left side, and we believe that our results could be reproduced using only one registration channel. We registered a lateralised status epilepticus pattern in two patients, which would probably have been identified by one biparietal registration channel. The greatest advantage of using two registration channels is that continuous aEEG registration can be guaranteed even if one electrode is loosened during the general nursing of the patient. We cannot exclude the possibility that localised EEG abnormalities, such as interictal epileptic activity, which would have been detected by conventional multichannel EEG registration, were missed because of the simplified two-channel technique we used. However, we believe that the use of a simplified technique with few registration channels is a prerequisite for successful aEEG-monitoring in the ICU setting.

The difficulty in predicting the outcome for comatose cardiac arrest survivors is well known, and while clinical

findings are still the best predictors [13], a multimodal approach has recommended [9, 10]. The usefulness of biochemical markers and of SSEP to prognosticate outcome after cardiac arrest and therapeutic hypothermia has been described previously [30, 31, 32], but none of the published methods has been as discriminative as the one presented here. This is the first report on the use of aEEG to predict outcome in adult comatose survivors of cardiac arrest who have been treated with hypothermia. Compared with conventional EEG, aEEG has the advantage of allowing continuous examination of the evolution of aEEG after transient ischaemia. Moreover, aEEG may be used to detect emerging, potentially treatable epileptic activity. We believe that aEEG will become an important prognostic tool for the early prediction of neurological outcome in hypothermia-treated comatose survivors of cardiac arrest.

Acknowledgements. This study was supported by the Swedish Research Council, grant No. 84 (Ingmar Rosén). We would like to thank Bodil Persson, EEG technician, for excellent technical support. The assistance of Gisela Railo, occupational therapist, Dr Tobias Cronberg and Dr Håkan Widner at the Department of Neurology, Lund University Hospital is gratefully acknowledged.

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